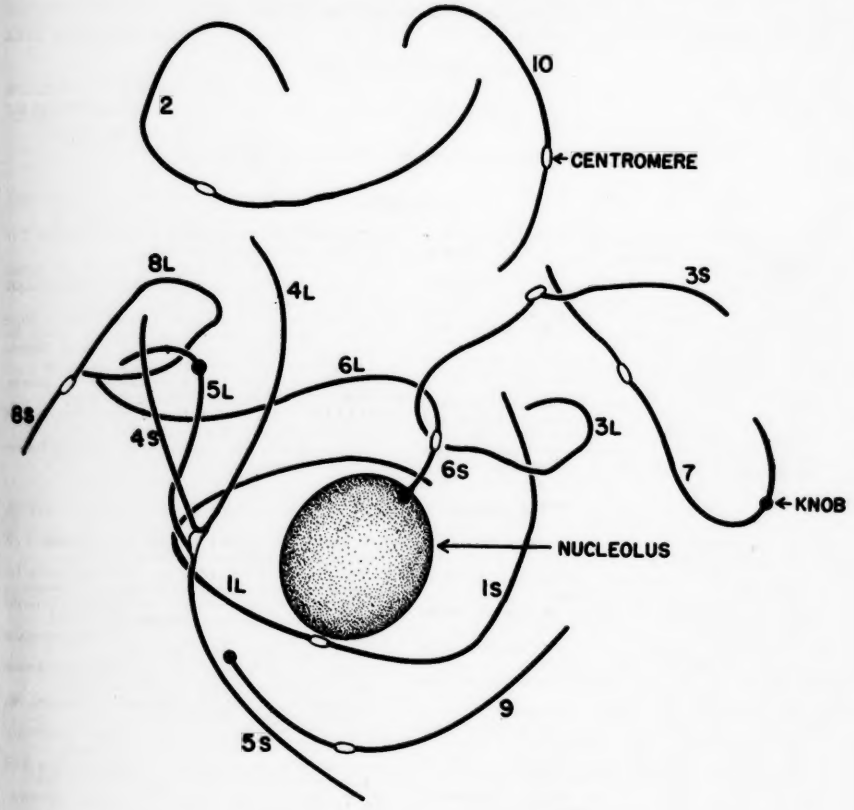
JUN 27 1950
S
PERIODICAL ROOM
GENERAL LIBRARY
UNIV. OF MICH.

The Journal of HEREDITY



THE TEN MAIZE CHROMOSOMES
(SEE PAGE 61)

Cyto-Genetics By Ear

Ringed Hair and Freckled Faces

A Four-Point Program for Gene Chasers

VOLUME 41 MARCH, 1950 NUMBER 3

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

Honorary Emeritus President, DAVID FAIRCHILD

MARCUS RHOADES, President — Vice-President, GEORGE M. DARROW

Honorary Vice-Vresident, W. E. CASTLE

RONALD BAMFORD, Secretary — Treasurer, SAMUEL L. EMSWELLER

Managing Editor, ROBERT C. COOK

Business Manager, VIRGINIA GLEIM — Assistant Editor, BARBARA LAKE

EDITORIAL BOARD

M. DEMEREC — RALPH W. PHILLIPS

H. J. MULLER — LAURENCE H. SNYDER — PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

O. S. AAMODT, Head Agronomist, Division of Forage Crops and Diseases, Plant Industry Station, Beltsville, Md.

RONALD BAMFORD, Professor of Botany, University of Maryland, College Park, Md.

GEORGE M. DARROW, Horticulturist in Charge of Small-Fruit Breeding, Horticultural Field Station, Beltsville, Maryland.

HAIG DERMEN, Cytologist, Division of Fruits and Vegetable Crops and Diseases, Plant Industry Station, Beltsville, Maryland.

SAMUEL L. EMSWELLER, Principal Horticulturist, Division of Fruit and Vegetable Crops and Diseases, Plant Industry Station, Beltsville, Md.

DAVID FAIRCHILD, Agricultural Explorer (Retired), U. S. Department of Agriculture.

H. J. MULLER, Professor of Zoology, Indiana University, Bloomington, Indiana.

HUGH C. McPHEE, Assistant Chief of Animal Husbandry Division, U. S. Department of Agriculture, Washington, D. C.

FREDERICK H. OSBORN, Deputy United States Representative on the United Nations Atomic Energy Commission, and on the United Nations Commission for Conventional Armaments.

MARCUS M. RHOADES, Professor of Botany, University of Illinos, Urbana, Illinois.

WILLIAM VOGT, Chief, Conservation Section, Pan American Union, Washington 25, D. C.

SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.

E. A. BESSEY, Professor of Botany, Michigan State College, Lansing, Michigan.

A. F. BLAKESLEE, Professor of Botany, Smith College, Northampton, Mass.

W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.

RUSSELL W. DUCK, Editor, *The Rural New Yorker*, New York City.

C. M. GOETHE, Sacramento, California.

HARRISON R. HUNT, Professor of Zoology, Michigan State College, East Lansing.

D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.

M. A. JULL, Professor of Poultry Husbandry, University of Maryland, College Park, Md.

C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.

H. H. NEWMAN, Emeritus Professor of Zoology, University of Chicago, (Clearwater, Fla.).

T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.

JOHN H. PARKER, Director, Midwest Barley Improvement Association, Milwaukee, Wisconsin.

JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.

A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.

GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.

EDMUND W. SINNOTT, Professor of Botany, Yale University, New Haven, Conn.

W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.

A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.

W. T. SWINGLE, Plant Physiologist (Retired), U. S. Department of Agriculture.

W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.

E. N. WENTWORTH, Armours' Bureau of Agr'l Research and Economics, Chicago, Illinois.

P. W. WHITING, Zoological Laboratory, University of Pennsylvania, Philadelphia, Pa.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 32nd Street and Elm Avenue, Baltimore 11, Md., and 1507 M St., N. W., Washington 5, D. C. Address all communications regarding editorial matters to 1507 M. St., N. W., Washington, D. C. Communications regarding the business management of THE JOURNAL OF HEREDITY may be addressed to the publication office, 32nd Street and Elm Avenue, Baltimore, Md., or to 1507 M St., N. W., Washington 5, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917 authorized December 29, 1918.

Contents copyrighted 1949 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$5.00 a Year. 50c a Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XLI MARCH, 1950 No. 3

CONTENTS

MEIOSIS IN MAIZE
By M. M. Rhoades 59

FOUR-POINT LINKAGE AS A GENETICS CLASS EXERCISE
By P. W. Whiting 71

A PEDIGREE OF GENERALIZED LENTIGO
By Alan C. Pipkin and Sarah Bedichek Pipkin 79

FOUR GENERATIONS OF RINGED HAIR
By Laurence M. Ashley and Robert S. Jacques 82

Notes and Reviews

A NEW TREATMENT OF GENETICS
By A. C. Fabergé and J. R. Singleton 67

HUMAN RACES AND THE SOVIETS
By R. Ruggles Gates 84

— URGENTLY NEEDED —

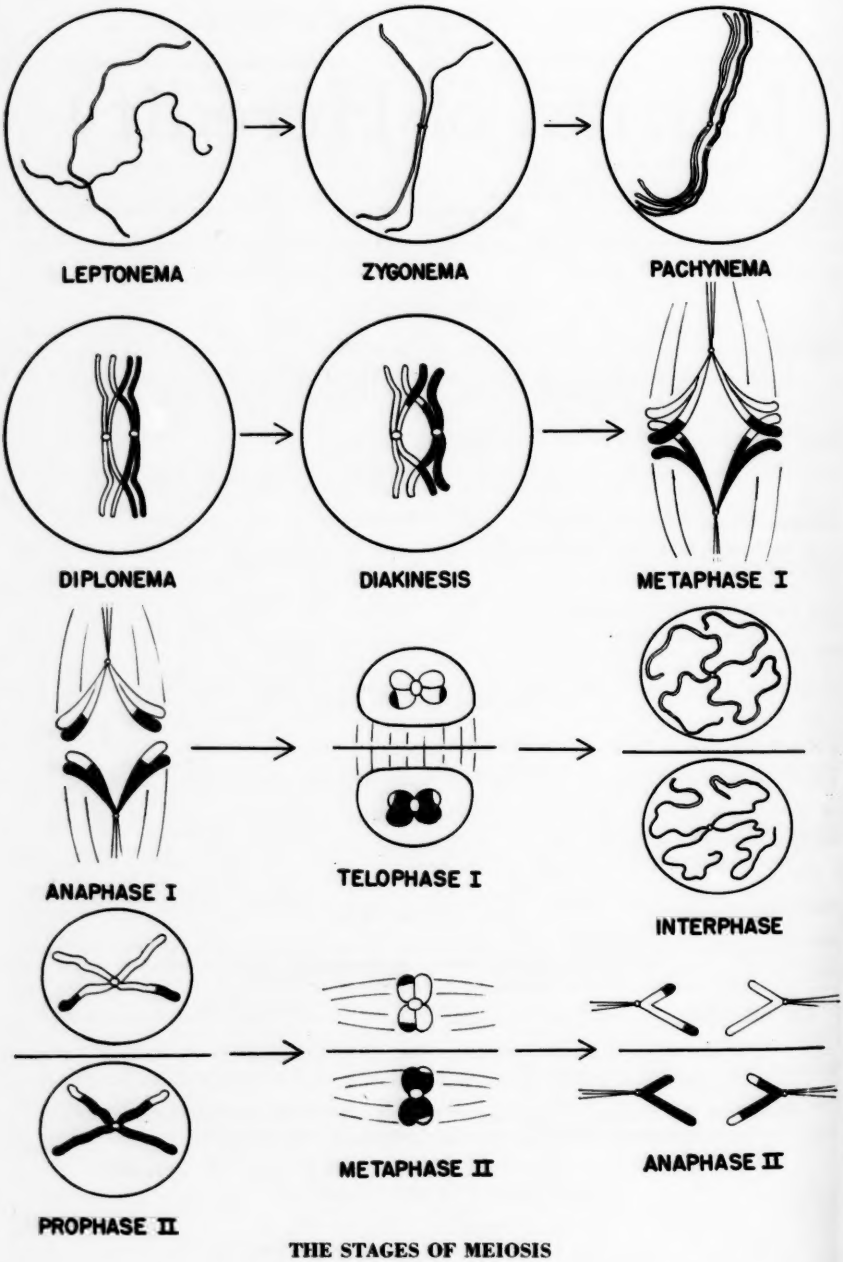
The following two issues of the JOURNAL OF HEREDITY are exhausted:

THESE ISSUES—OCTOBER 1945, FEBRUARY 1947— ARE URGENTLY NEEDED TO COMPLETE LIBRARY FILES.

Members will confer a favor by returning unwanted copies to us so that war-devastated libraries may be furnished with unbroken series.

WE WILL PAY $1.00 PER COPY FOR EACH OF THESE ISSUES OF THE *JOURNAL*.

Mail copies to
AMERICAN GENETIC ASSOCIATION
1507 M. Street, N. W. ***Washington 5, D. C.***



THE STAGES OF MEIOSIS

Figure 1

Highly schematized diagram of stages of meiosis in which a single pair of chromosomes is followed.

MEIOSIS IN MAIZE

M. M. Rhoades
Department of Botany, University of Illinois

ALTHOUGH more extensive cytogenetical studies have been conducted with maize than with any other plant, it is unfortunately true that no complete account of meiosis is available for the guidance of students and non-maize geneticists. That maize investigators have been somewhat remiss in not publishing a series of photomicrographs of the different meiotic stages is wholly understandable since the assembling of such pictures is a routine task. However when one has been engaged in teaching for several years, the need for a pictorial presentation becomes keenly felt; and it is to satisfy, to some extent at least, this need that this series of photographs is published in the Journal of Heredity. No claim is made of the excellence of these pictures; some of the stages of meiosis in maize, for example metaphase I, do not show the detail found in other forms; but on the other hand none of the stages is really bad and some are superb.

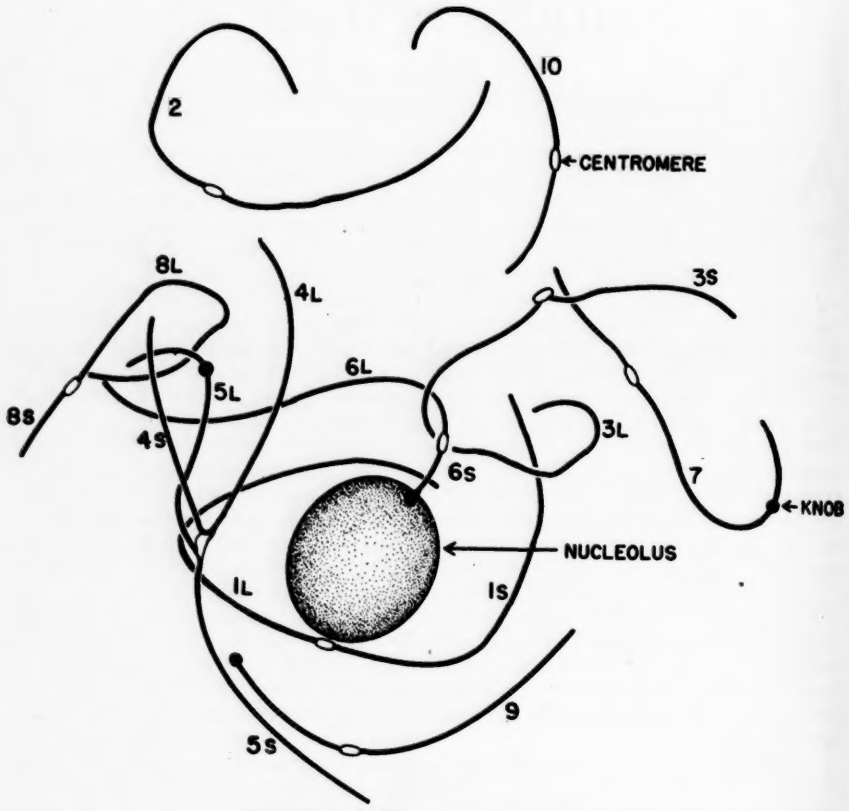
A diagrammatic sketch illustrating the important features of the various stages accompanies the photomicrographs. It is hoped that the two combined with the brief commentary, which makes no attempt at completeness, will make meiosis more understandable to the uninitiated. All of the photographs are from carmine or orcein smear preparations made in the writer's laboratory. Credit for the excellent pachytene figure should be given to Professor D. T. Morgan, Jr., of the University of Maryland, who made the preparation while a graduate student at Columbia University. In particular I should like to acknowledge my great indebtedness to the pioneering work of Dr. Barbara McClintock. The identification of the pachytene chromosomes and their association with specific linkage groups came almost entirely from her brilliant and illuminating studies. Maize cytogenetics surely would not occupy its present high estate were it not for her remarkable contributions.

In relating crossing over to chiasmata, and chiasmata to post-diplotene association, we have followed the generally accepted views. Likewise we have diagrammed the unpaired leptotene chromosomes as single, but it should be emphasized that unanimity does not exist about either the unsplit nature of the leptotene thread or the importance of chiasmata to metaphase pairing.

The early meiotic prophase stages (Figure 1) have never been adequately described and we have given only cursory attention to leptonema and zygonema. Photomicrographs of these two stages have not been included because of our failure to obtain satisfactory pictures. At leptonema the chromatin threads are extremely long and slender, being several times as attenuated as at midpachynema. Numerous small but distinct chromomeres may be clearly seen. The chromomeres visible at leptonema are more numerous and finer than at pachynema where the contraction of the chromonemata has brought previously separated chromomeres into juxtaposition. The conspicuous heterochromatic regions known as knobs, which are found at characteristic positions along the chromosome arms, are present at leptonema. Presumably the slender chromonema of leptonema is single and is devoid of coiling—a condition which would facilitate the gene-by-gene pairing which begins at zygonema.

Zygonema, at least in our smear preparations, is a difficult stage to analyze. The chromatin threads are drawn into a tight knot next to the nucleolus, but some pairing occurs before the so-called synizetic knot is formed. Occasionally a free arm protrudes from the chromatin mass, its paired nature revealing that synapsis has occurred.

After pairing is completed, the paired



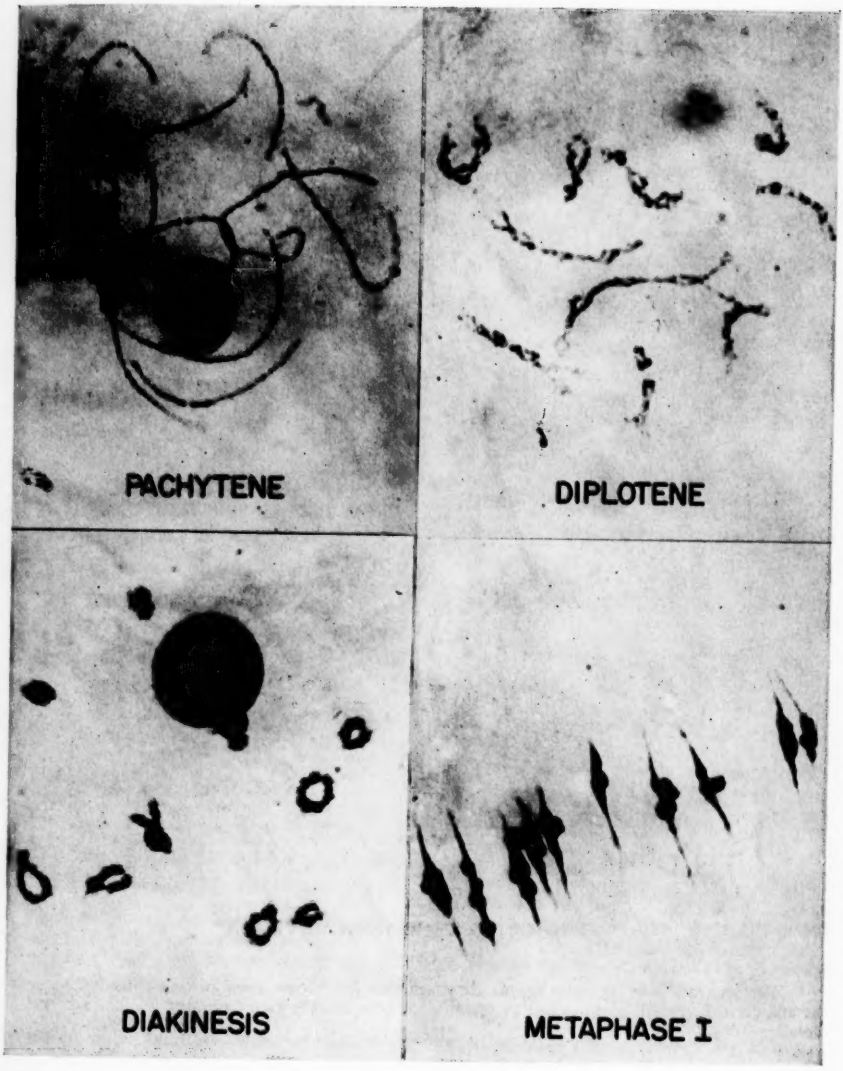
THE TEN MAIZE CHROMOSOMES

Figure 2

Schematic diagram of pachytene chromosomes shown in Figure 3. Each bivalent chromosome is represented by a single line in order to simplify the diagram.

homologues emerge from the chromatin knot. It is at pachynema that the great beauty of the maize chromosomes is most apparent and it is these chromosomes which have been so widely employed by many maize students in their analysis of structural changes (see Figure 8). The pachytene chromosomes are individually recognizable by their relative lengths, the distinctive chromomere patterns, the deep-staining knobs in characteristic positions, the position of the centromeres, and the deep-staining chromomeres adjacent to the centric regions. The nucleolar-organizing region of chromosome 6 remains associated with the nucleolus until its disappearance at the end of prophase. These chromosomes have been numbered according to their lengths—chromosome 1 is the longest and chromosome 10 the shortest member of the haploid complement.

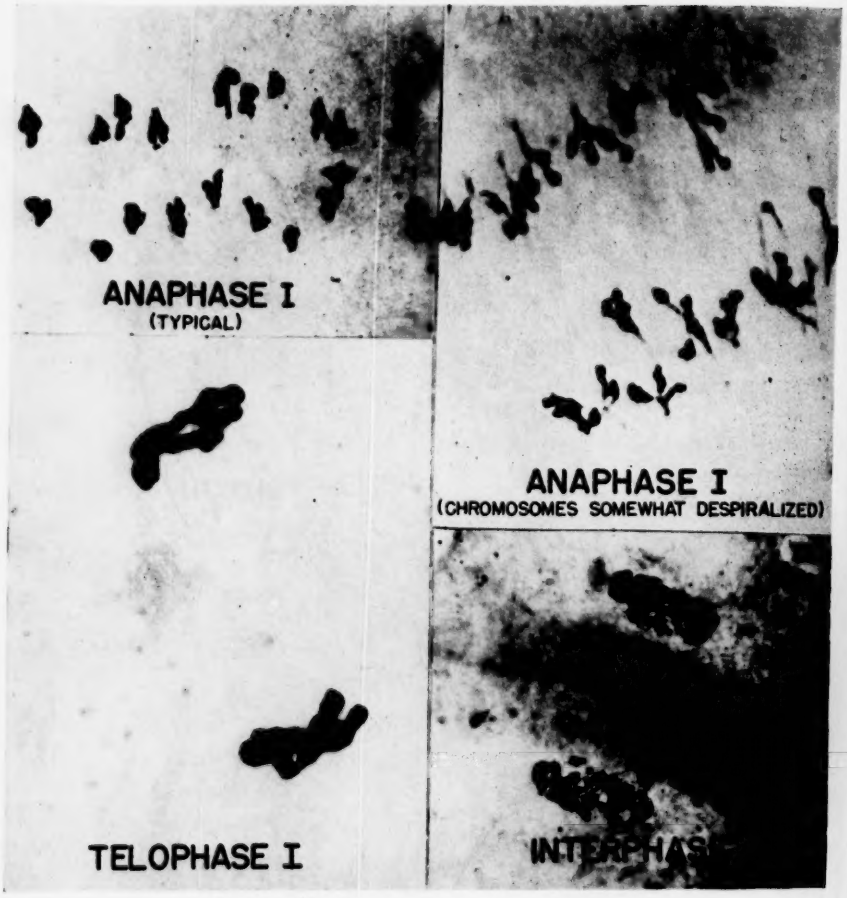
The centromeres of non-homologous chromosomes are often observed paired or stuck together at pachynema (see Figure 2) although they become free at late diplonema. Likewise the heterochromatic knobs on different chromo-



EARLY STAGES OF MEIOSIS

Figure 3

The earliest stages of the meiotic cycle, leptonema and zygonema, are not illustrated because good photographs of them have not been made. At pachynema the paired chromosomes are more contracted than during earlier stages. The homologous chromosomes are paired. The ten chromosome-pairs shown here are identified in the drawing on the opposite page.



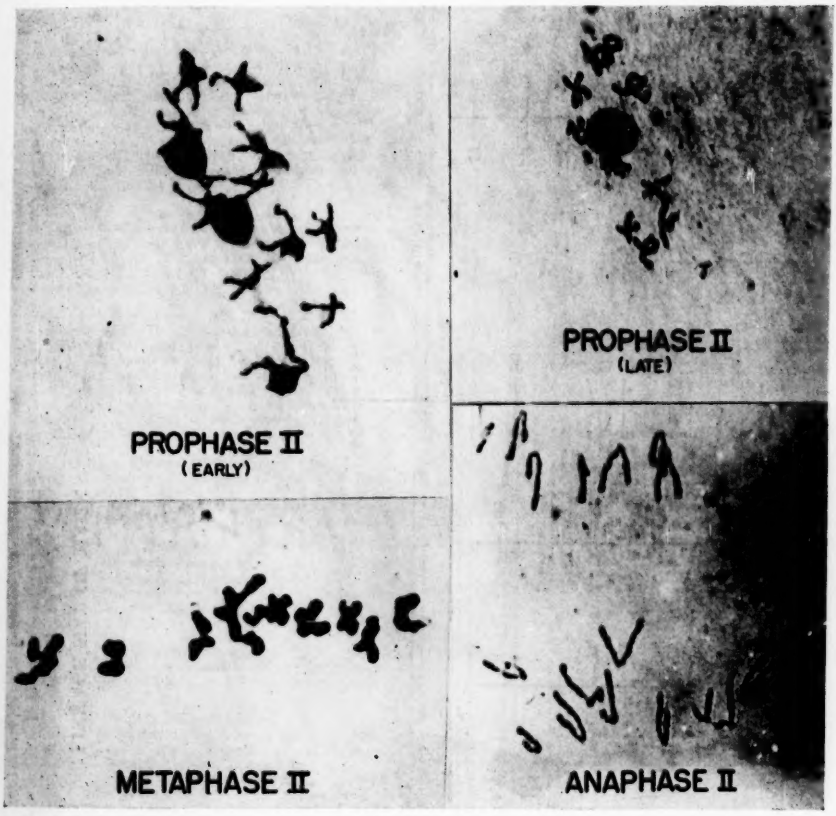
END OF FIRST MEIOTIC DIVISION

Figure 4

The separation of the homologous chromosomes (each comprised of two chromatids) occurs at anaphase I. A cell plate arises at telophase I between the two daughter nuclei. See text for details.

somes have a striking tendency to unite at pachynema. Presumably both the pairing of centromeres and knobs is due to the non-specificity of these regions—i.e. the centromere regions of the various chromosomes are of similar composition, as are the knobs.

The singleness of the leptotene thread has been questioned, but it is certain that the two paired homologues at pachynema each are divided into two chromatids, except for the centromere region of each homologue, which remains undivided until metaphase II. The pachytene pairs now may be designated as tetrads. The paired homologues are coiled relationally, but no unequivocal evidence of major or minor coils has been seen in my prophase preparations. Nevertheless the contraction in length, which is first



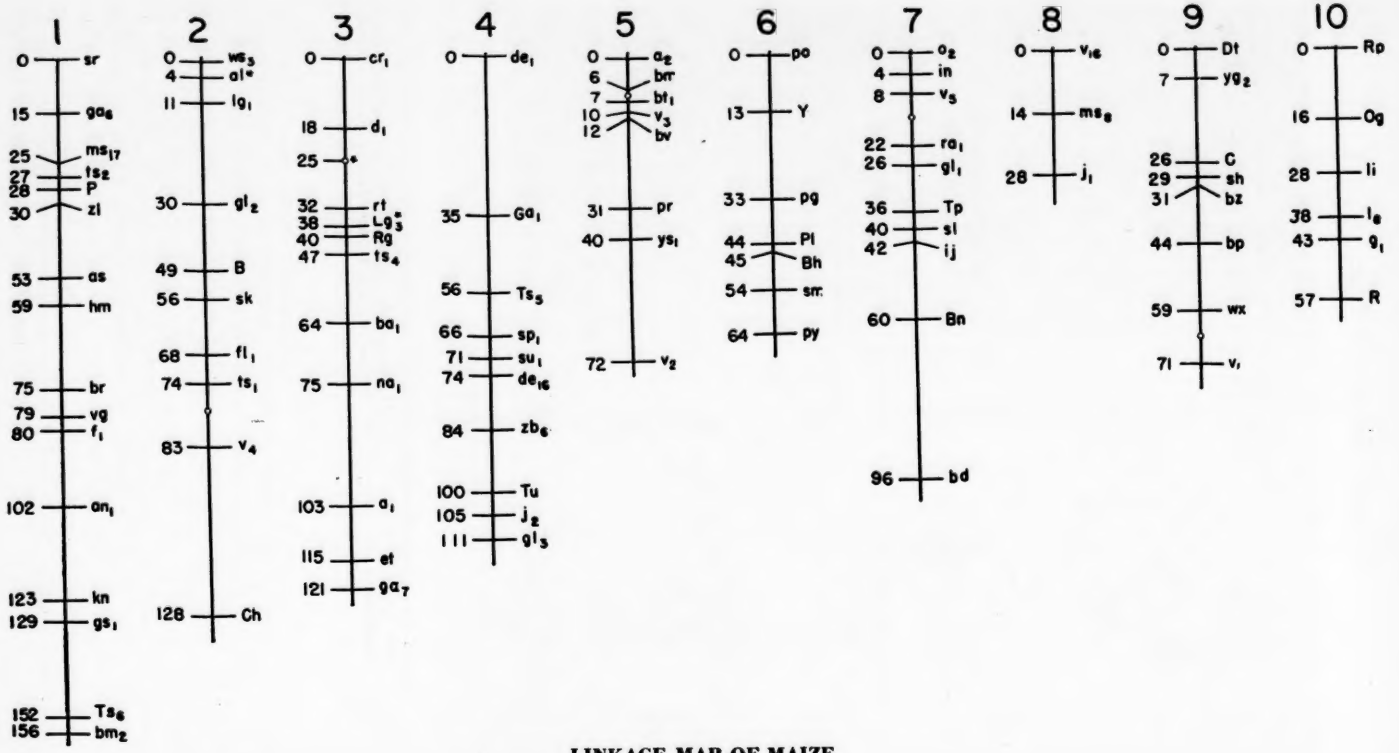
SECOND DIVISION

Figure 5

The long X-shaped dyads (two chromatids united by a common centromere) gradually condense until they become compact structures at Metaphase II. Orientation on the spindle then occurs, followed by centromere division and the separation of the two chromatids to opposite poles.

evidenced at pachynema and continues progressively throughout the meiotic prophase, is most likely due to a coiling of the chromatin threads or chromonemata since coiling of chromonemata has been observed in many diverse organisms. However, the possibility that some contraction may result from an actual loss of material from the chromosome has never been adequately explored. Some observations suggest that such may be the case.

Diplonema is characterized by a two-by-two opening out of the paired homologues to form loops and nodes. Most of the nodes represent chiasmata—i.e. an exchange of pairing partners—but it is not usually possible to follow the course of all four chromatids along the length of the chromonemata and it is possible that some of the nodes are due to a twisting of the paired homologues. The homologous centromere regions are usually associated at early diplotene but are



LINKAGE MAP OF MAIZE

Figure 6

The genes in linkage group 1 are carried by chromosome 1, etc. (See Figure 7.) The centromere regions whose map location has been established with some certainty are represented by circles. The zero ends of the maps are in the short arms of each chromosome. The location of those genes marked with asterisks is only approximate.

MAIZE CHROMOSOME ATLAS

Figure 7

Diagram showing relative lengths of maize chromosomes, location of knobs and centromeres (after Longley). The knobs (solid circles) are shown in their characteristic locations. Some strains of maize have chromosomes lacking in knobs while other strains have many knobs. The centromeres are represented by clear circles. To aid in the identification of the different chromosomes, the following comments call attention to important landmarks.

CHROMOSOME 1
Longest of complement. Knob or enlarged chromomere near end of short arm, and an enlarged chromomere at end of short arm. Average length at mid-pachytene is 82.40 micra. Ratio of long arm to short arm is 1.3:1.0.

CHROMOSOME 2
Sometimes difficult to distinguish from No. 5 but differs from the latter in ratio of arm lengths. The knob in long arm of No. 2 is further from the end than is the knob in long arm of No. 5. The regions adjacent to the centromere of No. 2 are more pycnotic than is the case in No. 5. Length is 66.50 micra at pachytene. Ratio of long:short arm is 1.25:1.0.

CHROMOSOME 3
Ratio of arms 2:1. Knob in long arm lies approximately in middle of arm. Some strains have a knob near the end of short arm. Length at pachytene is 62.00 micra. Ratio of long:short arm is 2.0:1.0.

CHROMOSOME 4
Knob or enlarged chromomere near end of short arm. Differentiated from No. 3 by ratio of arm lengths. Pachytene length is 58.78 micra. Arm ratio is 1.6:1.0.

CHROMOSOME 5
Arms of almost equal length. Knob in longer arm. Pachytene length is 59.82 micra. Arm ratio is 1.1:1.0.

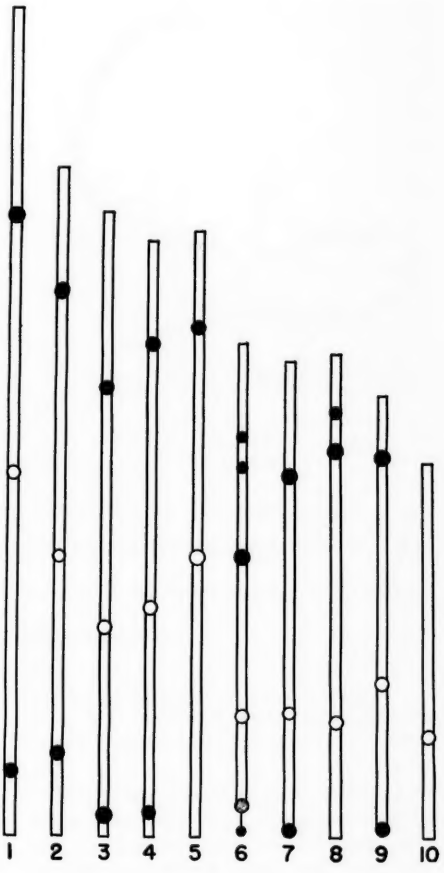
CHROMOSOME 6
Attached to nucleolus by deep-staining nucleolar organizing region. Length at pachytene 48.73 micra. Arm ratio is 7.1:1.0.

CHROMOSOME 7
Region of long arm adjacent to centromere is very deep staining. Occasionally a strain is found with a knob at end of short arm. Length is 46.78 micra. Arm ratio is 2.8:1.0.

CHROMOSOME 8
Marked disparity in arm lengths. Regions on both sides of centromere are pycnotic. Length is 47.48 micra. Arm ratio is 3.2:1.0.

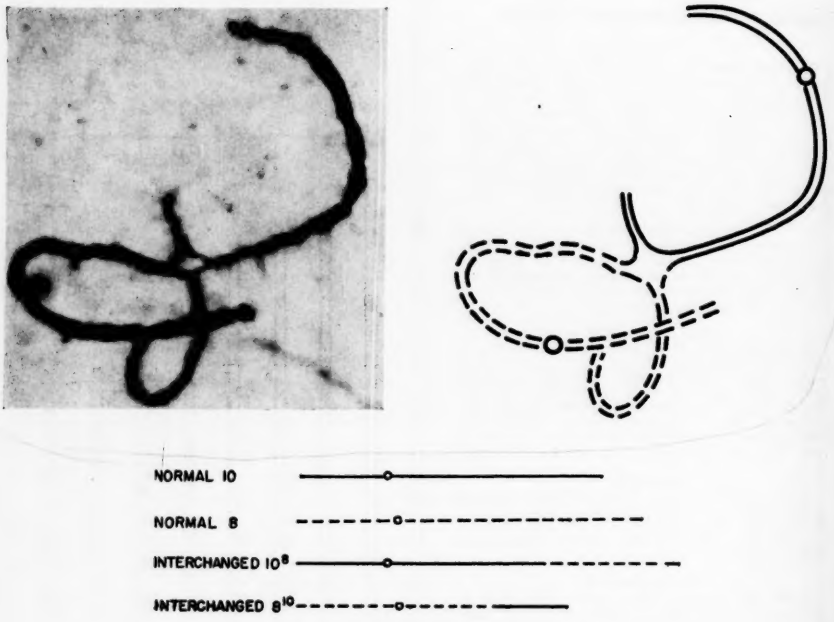
CHROMOSOME 9
Terminal knob at end of short arm. Pycnotic region occupying proximal third of short arm. Length is 43.24 micra. Arm ratio is 1.8:1.0.

CHROMOSOME 10
Shortest of complement. Short arm has distinctive chromomere pattern with deep-staining chromomeres next to centromere and small tapering chromomeres at end. Length is 36.93 micra. Arm ratio is 2.8:1.0.



The genes in linkage grouped by circles. The zero ends of the maps are in the short arms of each chromosome. The location of some certainty are represented by circles. The genes in linkage grouped by circles. The zero ends of the maps are in the short arms of each chromosome. The location of genes marked with asterisks is only approximate.

distinguishable from nodes by their differential staining. (The absence of the nucleolus in the diplotene photograph is due to the fact that this structure does not stain with orcein.) Most geneticists believe that each chiasma represents a genetical crossover and the diagrams have been drawn in accordance with this concept. Since there is little movement of chiasmata towards the free ends during diplonema (a process known as terminalization), diakinesis and metaphase I find some bivalents with interstitial chiasmata, although most of the chiasmata are near the ends. This concentration of chiasmata towards the ends may be caused in part by terminalization but is probably related to the more frequent occurrence of crossing over in distal regions.



CHROMOSOME INTERCHANGE

Figure 8

Photomicrograph and camera lucida drawing of a heterozygous translocation involving chromosomes 8 and 10. The two normal and the two interchanged chromosomes are diagrammed to the right of the photomicrograph.

At the close of diakinesis the nucleolus disappears as its substance enters the chromosomes and contributes to the matrix of the deeply staining metaphase chromosomes. The nuclear membrane breaks down, the spindle is formed and the tetrads are oriented on it with the two homologous centromeres of each tetrad lying on opposite sides of the equatorial plate. The half spindle fibers elaborated by the centromere at metaphase I are concerned with the poleward movement of the chromosomes. The appearance of the tetrads at metaphase I is determined by the number and position of chiasmata in the two arms.

The separation of the tetrad into its two pairs of chromatids (dyads) begins with the anaphase movement. As the centric region leads the way to the poles, the chiasmata which were present at metaphase are unraveled; the four arms of each dyad are passively pulled along to the pole. Dyads at anaphase I appear as double V's if the centromere is near the middle while one with a more terminally placed centromere resembles a double J. In either case the apices of the V's and J's are conjoined at the undivided centromere. The four arms of a dyad do not lie closely appressed during the poleward journey but diverge as if they were mutually repelling each other. The first division is reductional for the centromere regions, and for those regions in each arm between the centromere and the first chiasma. Regions distal to the first chiasma undergo an equational first division.

In mid-anaphase the matrix material which invests the chromonemata swells to form a light staining region in which

the deeper staining chromatin is visible. By telophase the four arms of a dyad are much contracted; in many cells each arm is a spherical mass of chromatin. The matrical material from the various dyads coalesces as they form a compact group at the pole. The chromonemata elongate (uncoil) during interphase. A typical metabolic nucleus is not formed, although the anastomosing of the matrix material results in considerable dispersion of the chromonemata. The cell plate, arising at telophase, divides the mother cell into two daughter cells, each with ten dyads.

At early prophase II the long X-shaped dyads are each embedded in the confluent light-staining matrical material. In later prophase stages the chromosome arms become progressively shorter and a reduction in the amount of visible matrix material occurs. The dyads become increasingly deeper stained as the matrical substance becomes incorporated into the chromosomes. By metaphase II no matrix material is visible, the dyads are compact X-shaped structures which resemble those found at telophase I save that in the latter the matrical material loosely invests the chromatin. The open nature of the X-shaped dyads indicates a strong repulsion between homologous arms.

Prior to metaphase II the nuclear membrane disappears. A spindle develops on which the dyads become oriented with the still undivided centromeres lying in the equatorial plate. The centromere of each dyad now becomes functionally double, spindle fibers are formed by each of the two chromatids and a poleward migration commences.

The anaphase II chromosomes are single V's or J's as contrasted with the double V's and J's of anaphase I. At anaphase II the matrical material about each chromosome begins to swell and at telophase II the chromosomes at each pole are joined by this anastomosing material. A cell plate is formed between the daughter nuclei, and the second meiotic division is now completed. Each of the four chromatids comprising the pachytene tetrad has been distributed to one of the four haploid cells formed as a consequence of the two meiotic divisions.

The above account describes the course of microsporogenesis in maize. Megasporogenesis has received relatively little study but no difference in the behavior of the chromosomes has been noticed. One notable difference between the formation of pollen and ovules is that the former process produces four spores all of which are functional, while megasporogenesis gives rise to only one functional spore.

The diagram of the relative lengths and diagnostic characteristics of the ten chromosomes, together with the linkage maps, may be of value to those who plan to study the cytogenetics of maize.

In order to preserve some continuity to the above account of meiosis, I have refrained from citing published work in which meiotic phenomena have been described. I have drawn rather heavily upon certain publications of my colleagues — namely those of Longley, Burnham, Randolph, Beadle, Anderson, and others, but above all on those by McClintock. It would, however, be unfair to hold them responsible for any mis-statements which may have been made.

A NEW TREATMENT OF GENETICS

*THE Elements of Genetics** is, say the authors in their preface, "the first attempt to represent the whole scope of genetics, the whole of what has always been needed." Drs. Darlington and Mather have focused their attention

*Darlington, C. D. and Mather, K., *The Elements of Genetics*. 1949. London: Allen and Unwin. New York: MacMillan, pp. 446, 88 figures, 31 tables $3.75.

successively on three major levels. Part I, Individuals, considers the main body of classical genetics, Mendelism, chromosome behavior, continuous variations and the consequences of changes in the genetic systems. Part II, Cells, is essentially an attempt to relate the genetic systems of the nucleus to the general cellular environment and to establish order among the various non-nuclear genetic elements. In Part III, Populations, the facts, concepts and conjectures derived from study of smaller units are applied at the level of the population. Both authors have clearly made contributions to all three sections, but these contributions differ so radically in approach, in content, in style, and particularly in methodology there is generally no difficulty in distinguishing them. It is proposed to review them separately.

Readers will remember the many violent attacks to which Dr. Darlington's *Recent Advances in Cytology* and other works were subjected. To avoid a repetition, this author has prefaced the present volume with a most disarming preface, in which warning is given that *Dangerous Speculations* must be expected.

Now the reviewers, doubting whether scientific speculations are ever dangerous, turned to the book with high expectations. In physics, for instance, long range speculation is a perfectly honorable method, and has, as everyone knows, given remarkable results. Only there is a rule in this game: The other fellow must know when you are speculating and when you are giving facts. Thus Niels Bohr brillantly *speculated* that the angular momentum of electrons in their orbits can only take exact values which are simple multiples of $h/2\pi$. Calculation then shows that the hypothetical permitted frequencies are in fact exactly the *measured* wave-lengths of the hydrogen spectrum. *Data* and *hypothesis* are confronted, they are not confounded. The importance of proceeding in this way is particularly apparent in this case, where the next important step forward came from considering cases which do not fit the rule, and the rule itself then proved to be but a partial truth.

We carefully searched through Dr. Darlington's sections for such genuine hypotheses, but in vain. They may be there, but if so they cannot be found, so inextricably are they mixed with facts, with near-facts, with observations both published and unpublished and with mere obscurities and extravagancies of literary style. The situation is made worse by the logic used, which is of a kind more proper to the law court than to science, rigor and precision being sacrificed to the one purpose of proving Dr. Darlington right. Belling once proposed that Darlington distinguish his conjectural propositions by a (C) placed hard by. In the present volume it might have been more economical to distinguish the unarguable facts and observations by (F).

Cytology has special difficulties of its own, unique in degree if not in kind. Its very observations are heavily loaded with the observer's bias, and infinite precautions are in order. Darlington's reaction to this situation has always been superficially one of poised assurance, fundamentally one of despair. Why try to be objective if it's so hard?. . . But genetics can surely claim an objectivity rare in biology and here such an outlook is worse than inexcusable. To return to our analogy, spectroscopists have some right to say they can *predict* H wavelengths, but they do not on this account permit themselves liberties in *measuring wavelengths* or in *tabulating* spectral lines.

Let us turn to some examples, and let it be clear they are not exceptional. We had the embarrassment of choosing them from among a great many.

A. "Only in abnormal individuals, or under abnormal conditions, as from sudden changes in temperature, do we find the chromosomes breaking up. . . ." (p. 100).

B. "Between the two types of hybrid, in which structural hybridity is too slight or too great to be defined, lie a host of crosses between species like those in *Lilium* or *Drosophila* in which the chromosomes of the parents differ recognizably by inversions, interchanges, duplicated fragments, or more complex changes.

Allopolyploids are derived from hybrids of all such types and by successive additions appear as tetraploids, hexaploids and so on" (p. 139-140).

"In experiments healthy and vigorous polyploids can readily be produced in *Drosophila* (p. 99).

"Both these plant hybrids recover fertility when the number of their chromosomes is doubled; whereas tetraploidy leads to no such recovery in male flies" (p. 231).

C. "The uncoiling of chromosomes at telophase depends on their throwing off this coat of nucleic acid. How do they do this? Evidently the nucleic acid is depolymerised and broken down. At the same time the products of the action of the genes, now uncovered, fill the nucleus . . ." (p. 146).

"Their X and Y chromosomes suffer from a nucleic acid surfeit and one which is greater at a lower temperature. In consequence of the surfeit they fail to pair. . . . The Y chromosome, which, as we know, is still more largely heterochromatic than the X, is so heavily flooded with nucleic acid that it fails to divide at all and is lost" (p. 337).

Now these are all very remarkable statements. In no case are any citations of literature given. Left to their own devices, the reviewers suspect that *A* may involve unpublished observations of the authors. The reviewers would like to know, for one of them has made comprehensive experiments on the production of chromosome breaks by temperature shocks, with completely negative results. *B*, we think, is merely bad syntax; the reader would inevitably conclude, however, that tetraploids and even hexaploids can be currently obtained in *Drosophila* and there is nothing elsewhere in the book to contradict this. *C* we would attribute to somewhat excessive embellishment of verbal expression. But why should the reader be left to make these guesses? It is after all, conceivable that Dr. Darlington has discovered a means of raising polyploids other than triploid in *Drosophila* or that he has by some device seen the products of gene action fill the nucleus.

C is, of course, typical of most of Dr. Darlington's writings. Readers were at first shocked and pained, but with the passage of years their taste was educated. Indeed they began to open Dr. Darlington's new publications with anticipation and were often handsomely rewarded. These irreverent amateurs will find the present book of interest: They will, among many things, read about chromosomes in the language of internal ballistics, about the "nucleic acid charge" and about a centromere which ". . . explodes into useless fragments" (p. 104).

Dr. Mather's contributions are altogether more pedestrian and can be reviewed as one would normally review a scientific book. These contributions are in the fields of elementary statistics, quantitative inheritance, populations, mating systems, natural selection and to some extent speciation and a few other topics.

In chapters 3 and 4 will be found an elementary account of the chief statistical concepts and of quantitative inheritance. These expositions are admirably lucid and, despite their brevity, remarkably complete. This is no small achievement, for in these fields most expositions have been notorious for their obscurity. The author is not so fortunate when he departs from the realm of artificial models. The experiments he describes are often inadequate in scope and lack any serious attempt at good statistical control, the very subject he so admirably expounds elsewhere. This is particularly true of the *Drosophila* experiments, where we are sorry to see no mention of the much more extensive work of Payne, done thirty years ago.

Most of Dr. Mather's treatment of quantitative inheritance follows strictly orthodox lines, and his principal contribution is a more adequate consideration of linkage. He does appear to believe, however, that the genes involved in quantitative inheritance are special not only by virtue of their effect, but differ in a more intrinsic way from the ordinary large effect genes, and even goes so far as to postulate that they are largely (or entirely) located in heterochromatin, having some particular relation to nucleic acid. It does not seem to us that the experiments published to date support this view which, on *a priori* grounds, seems implausible. We partic-

ularly regret the reintroduction of the term "polygene" to emphasize this supposed intrinsic difference. Much of this concept and even the word "polygene" have been taken from a rather bad and almost forgotten German textbook published in 1913 (Plate, L., Vererbungslehre, Leipzig). In those days such confusion was excusable, but it is amusing to note that in the same year Johannsen attacked the idea with some violence and for reasons which are largely valid today (Elemente der Exakten Erblichkeitslehre, 2nd Ed., Jena).

It seems clear that yield, height, etc., in fact depend on the most diverse physiological and developmental processes, and are therefore, we believe, determined by the most intrinsically diverse genes—genes which have just this in common, that they happen to affect height, yield, etc. It is best therefore, to speak simply of quantitative inheritance and of height or yield determining genes, as has been done for the last forty years. If there is need for a special adjective, the word multifactorial should be used. While it may lack phonetic elegance, it is not loaded with undesirable implications. Geneticists have learned to live with much worse phonetic monsters.

The description and discussion of mating systems is in general very clear and complete. It is curious to read (p. 260) "Outbreeding, so far as we know, never supervenes on inbreeding," for there would seem reasonable grounds to suppose that this step happened in the evolution of fungi, for example. On page 247 will be found an ingenious graph which clearly summarizes the genetic properties of various incompatability systems in fungi, and places their comparison on an orderly basis.

The discussion of those initial divergencies in populations which lead to speciation is remarkable for its complete omission of any mention of genic drift, the so-called Sewall Wright effect and the properties of small populations. Instead, discontinuities not due to simple mendelian segregation but to other chromosomal effects are emphasized, such as inversion, translocations and the breakdown and recombination of linked complexes. It is perhaps a good thing that attention should be drawn to their widespread importance.

In so far as the body of classical genetics and cytology is concerned, the book contains few, if any, new facts or concepts. Nor does it contain an adequate selection of the old ones. It would seem, for example, that since such forms as the coccids (and *Luzula*, among plants) do not fit Dr. Darlington's beautiful schemes, these organisms had been relegated to the outer limbo of forgotten things. At least, no mention of them is made. Upon reading the section devoted to the relationships between the genetic determinants and the cellular environment, the reviewers were afflicted with qualms and misgivings, amounting at times to nausea. The urge to explain all phenomena in terms of nucleic acid, a tendency which permeates this region of the book, is probably a result of the somewhat mystical properties with which some geneticists have endowed the substance. Luckily, Dr. Darlington is sufficiently vague on these topics to escape being formally incorrect.

It is a heavy responsibility to say that a given book is harmful: that the sum of good in it is outweighed by the evil. How can one ever know? Of the present volume we will only say this: That it will give a distorted picture of genetics to other scientists who might happen to seek information therein, and a disastrous misrepresentation of the logical standards of other geneticists. A teacher who might wish to give this book to his students will have to prepare for them a list of errors, exaggerations and, shall we say, *non-facts*, a list having not far from one entry for each page of the book.

A. C. Fabergé
J. R. Singleton
University of Missouri

FOUR-POINT LINKAGE AS A GENETICS CLASS EXERCISE

P. W. WHITING
University of Pennsylvania, Philadelphia, Pa.

FUNDAMENTAL to an understanding of cytogenetics is the principle that genes are arranged in linear order on the linkage map. A three-point test illustrates this but a four-point test is more convincing, because in the former it might just happen that the three points were in line instead of at the angles of a triangle. Moreover, with four points one can have triple crossovers which must be added to the doubles in calculating coincidence and, along with the proper doubles, to the three singles in obtaining the three map distances. Four points involve these principles so that it is then easy for the student to go on to five and six points, while three-point work is inadequate. These facts stimulate the imagination of the better students and lay the foundation for an understanding of chromosomal irregularities.

Recently the writer has been developing the four-point idea in his classes in genetics . Working through a four-point test in an hour examination has become a *sine qua non* for passing the beginning course. The student is given the frequencies of the sixteen genotypes obtained from the test cross (or more usually the numbers of the sixteen types of males obtained parthenogenetically from the tetraheterozygous virgin females of Habrobracon). He is told from which of the two parents each mutant gene is derived, but he knows nothing of the arrangement of the genes along the map. Routine steps which must be memorized are outlined on mimeographed forms supplied to the class because the subject is inadequately treated in the textbooks. These steps are discussed in the lectures and serve to avoid loss of time and hit-or-miss guessing in making up the map.

Those taking the laboratory work are supplied with two stocks of Habrobracon from one of which they must breed and isolate virgin females to be crossed with males bearing the other traits. Female-sterile traits or combinations difficult to run homozygously are introduced on the male side from sons of heterozygous mothers. The F_1 tetraheterozygous females are then reared and isolated as virgins. Each student is required to breed at least five hundred F_2 haploid males and to classify and separate them into sixteen vials of alcohol each of which should contain wasps of a single genotype. These vials are passed in with the written report at the completion of the semester's work (two semester credits).

All the students do the same experiment in any one semester, so that it is necessary to have only two stocks developed for distribution. Instruction is given in technique of rearing, isolating virgins, etc., at the beginning and the students are permitted to put in as much time as they find convenient. Some may then finish three or four weeks before the semester's end.

There is relatively little of the instructor's time required after the preliminary information is given and before the reports and specimens are turned in at the end. It is up to each student to do the work and the dishonest student has no chance of faking the counts since the specimens must be produced properly classified. Some students have "green fingers" and get good numbers easily. Others have the "lethal touch" and must compensate by hard work. One girl, at first in the latter category, put in endless hours with no tangible results during the first half of the semester. Then she acquired the tricks and finished with greater numbers than anyone else in the class, an A+ grade and a high enthusiasm to take further work in genetics.

It costs the instructor about one and one-half hours of time at the end to check the report and specimens of each student. The report is first checked with the aid of a Munroe calculator. Then each vial of specimens is poured into a Petri dish and the wasps examined and counted. Correction is made on the report. From the corrected reports the laboratory grades are then made up. All are on the same basis since all do the same experiment, classifying the same traits. It frequently happens that the written report would rate an A grade, but that errors are shown when the specimens themselves are compared, thus lowering the grade.

The wasps from any one experiment are kept in a single vial and are used as laboratory material in short courses which do not allow time for breeding specimens. A little glycerine is added to the alcohol to prevent drying and the wasps are placed on a glass plate, brushed out into a row and separated into classes under a binocular. Most of the traits are as easily recognized as in the fresh material and the same collection may be used repeatedly.

A Four Point Cross

Four traits recently tested have been stubby antennae, *sb*; black body color, *bl*; lemon body color, *le*; and cantaloup eye color, *c*. The two body color differences illustrate factor interaction and the four possible genotypes may be recognized without difficulty. Wild type is honey yellow with black markings; lemon is bright lemon yellow with restricted black markings; black has black markings much extended and the limited yellow areas reduced to buffy; black lemon differs from lemon in being greenish yellow with sooty suffusion.

There are sixteen different ways in which four mutant genes may be introduced into a cross (the coefficients of the fourth power of the binomial, 1:4:6:4:1) but since half of these are reciprocals of the others there are only eight *different types* of tetraheterozygous F_1 females. (Figure 9) Mutant genes derived from the mother (P_1 ♀) may then be none, one combination; one, four combinations; two, six combinations; three, four combinations; four, one combination. The binomial theorem may similarly be applied to five-, six-, etc. point crosses.

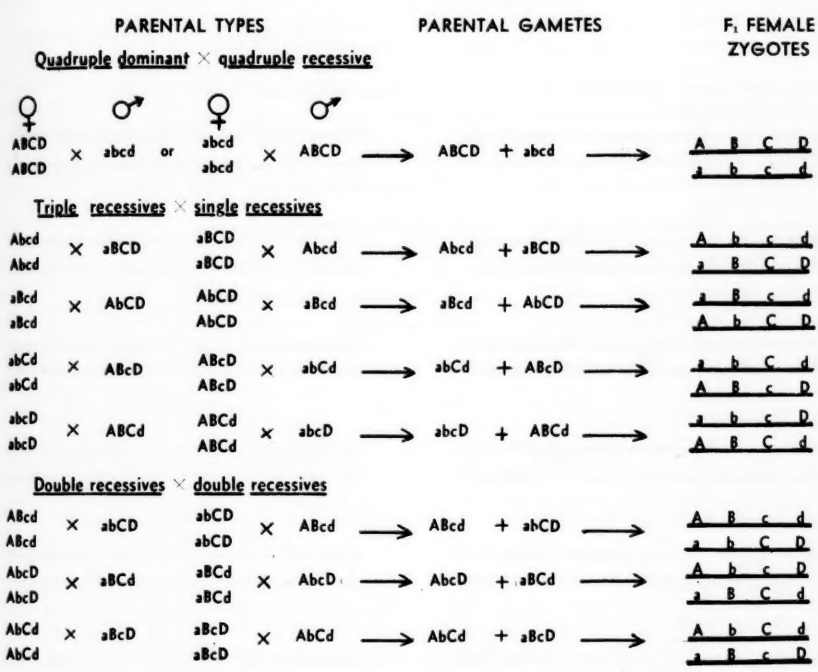
Sixteen Possible Types of Chromatids from Tetraheterozygous Females

There are sixteen possible types of eggs laid by females heterozygous for four linked genes. Each type, containing its characteristic chromatid, develops parthenogenetically into an azygote, a haploid male, phenotypically distinguishable from the others (Figure 10).

The numbers of types of males carrying increasing numbers of recessive genes correspond to the coefficients of the *fourth* power of the binomial, $(a + b)^4$: 1 carrying no recessive; 4 types with one; 6 types with two; 4 types with three; and 1 type with four (Figure 9).

There are *three* crossover regions. The numbers of types of crossing over, therefore, correspond to the coefficients of the *third* power of the binomial, $(a + b)^3$:1 straight or non-crossover; 3 singles; 3 doubles; 1 quadruple. Each of the eight types of crossing over produces two of the sixteen possible types of chromatids. However, because crossing over occurs in the four-strand stage and may involve more than two chromatids, alternative types of chromatids are not always formed in a given tetrad. Moreover, only one strand is recovered, retained in the egg after meiosis. The diagram (Figure 10) expresses what is to be expected *on the average* and is adequate for the present purpose. It would not be adequate for tetrad analysis work as in Neurospora.

In tables 1-5 the symbols for the wild type alleles are not expressed but these wild type alleles are of course understood to be present in the absence of the mutant factors *sb bl le c*. The members of each pair of alternative chromatid types are placed on the same line in the two


PARENTAL TYPES
PARENTAL GAMETES
F1 FEMALE ZYGOTES
Quadruple dominant × quadruple recessive
♀ ♂ ♀ ♂
ABCD / ABCD × abcd or abcd / abcd × ABCD → ABCD + abcd →
Triple recessives × single recessives
Abcd / Abcd × aBCD
aBCD / aBCD × Abcd → Abcd + aBCD →
aBcd / aBcd × AbCD
AbCD / AbCD × aBcd → aBcd + AbCD →
abCd / abCd × ABcD
ABcD / ABcD × abCd → abCd + ABcD →
abcD / abcD × ABCd
ABCd / ABCd × abcD → abcD + ABCd →
Double recessives × double recessives
ABcd / ABcd × abCD
abCD / abCD × ABcd → ABcd + abCD →
AbcD / AbcD × aBCd
aBCd / aBCd × AbcD → AbcD + aBCd →
AbCd / AbCd × aBcD
aBcD / aBcD × AbCd → AbCd + aBcD →

EIGHT TYPES OF TETRAHETEROZYGOUS F_1 FEMALES

Figure 9

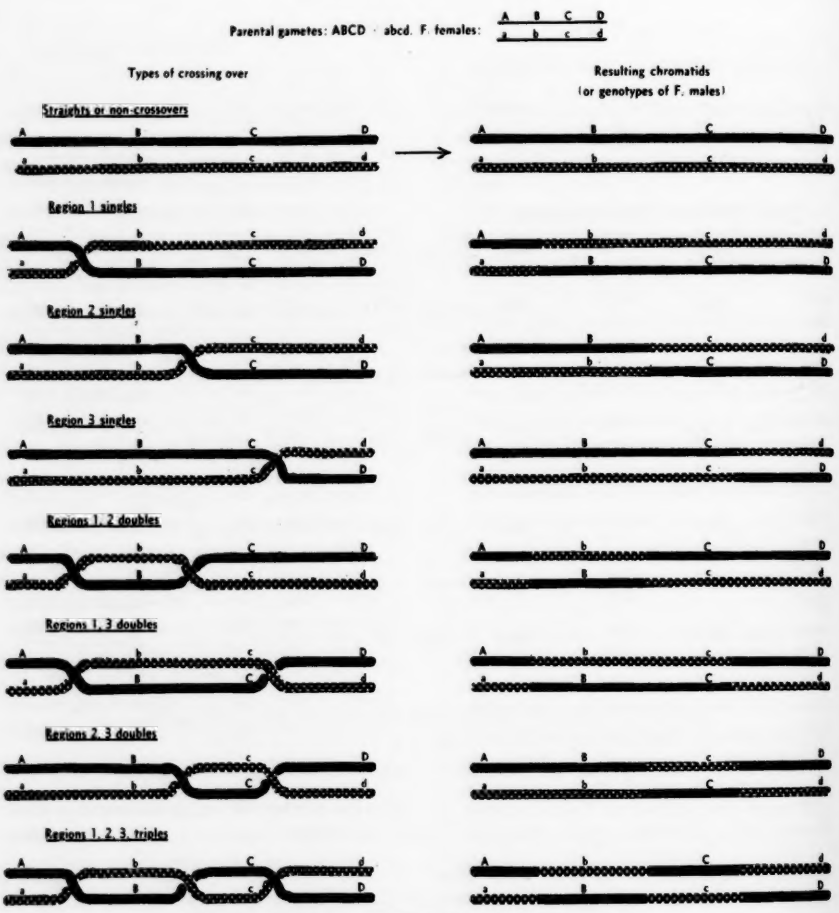
In a cross involving four pairs of genes (linked or independently inherited), there are sixteen different crosses of homozygous P_1 types. (In wasps the males are haploid, but the same reasoning applies to diploid males). These eight direct and eight reciprocal crosses produce eight different types of tetraheterozygous F_1 females.

different columns. The reader may substitute these mutant gene symbols, *sb bl le c*, for *a b c d* respectively, using either + + + + or *Sb Bl Le C* for the dominant alleles. Figure 11 shows how in each cross a particular phenotype represents a different type of crossing over.

A total of 6,148 F_2 males were classified from wild type females crossed with *sb bl le c* males (Table I); of 10,042 from *sb* females by *bl le c* males (Table II); of 3,362 from *le* females by *sb bl c* males (Table III) and of 4,670 from *c* females by *sb bl le* males (Table IV). The remaining experiment with one factor from the mother, namely *bl* females by *sb le c* males, has not yet been carried out. Clark[2] (p. 63) counted 6,571 F_2 males from *bl c* females by *sb le* males (Table V). Two more of the six possible combinations of the two genes from the mother may be tested in the future. The others are reciprocals.

Working Out the Problem

Steps taken by the students in a quizz problem may be illustrated from the data of Table IV. A laboratory report based on their own experiments follows the same system. The students are told that the mother is cantaloup and that the father is black, lemon, stubby (for example), so that no hint is given of the arrangement of the genes on the map. Frequencies of the sixteen classes of F_2 males are given and also their grand total, 4,670, so that the students may have a check and save time in arithmetic. The students must list the alternative pairs of genotypes in two columns with their frequencies headed by the pair of


Parental gametes: ABCD · abcd. F. females: A B C D / a b c d
Types of crossing over
Resulting chromatids
(or genotypes of F. males)
Straights or non-crossovers
Region 1 singles
Region 2 singles
Region 3 singles
Regions 1, 2 doubles
Regions 1, 3 doubles
Regions 2, 3 doubles
Regions 1, 2, 3, triples

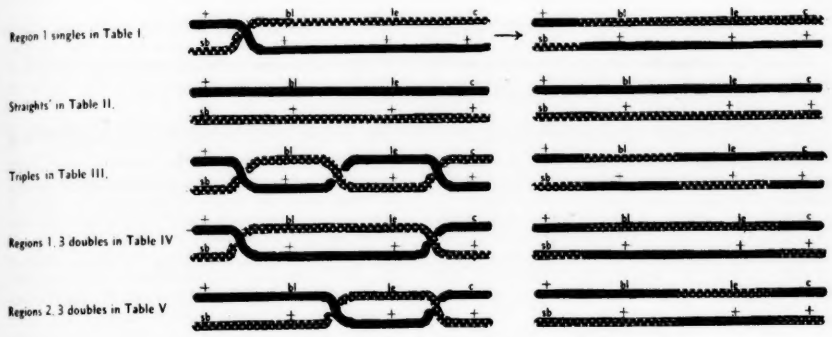
SIXTEEN DIFFERENT F_2 MALE GENOTYPES

Figure 10

F_1 females tetraheterozygous for four linked genes produce sixteen different combinations of dominant and recessive alleles in their gametes. These correspond to sixteen distinct phenotypes of haploid males. The cross shown here represents the F_2 of the quadruple dominant by the quadruple recessive. Other P_1 combinations give the same sixteen F_2 male types, but the classification as crossover types is different. Only two of the four chromatids are shown. See page 72.

straight* classes. The others are set down in general in descending order of frequency. Thus the triples are likely to be at the bottom of the column and there is a fair chance that the singles may precede the doubles. From mothers heterozygous for four genes there are to be expected sixteen types of eggs,—one pair of straights, three pairs of singles, three of doubles and one of triples. From pentaheterozygous mothers there would be thirty-two types of eggs,—one pair of straights, four of singles, six of doubles, four of triples, and

*In an exceedingly clear and concise article Bridges[1] has discussed the terms crossing-over, map distance and recombination. These words are frequently confused in the textbooks which often use crossovers as synonymous with recombinations, thus bewildering the students. No convenient terms have come into general use in the research literature to designate non-crossing-


Region 1 singles in Table I.
Straights in Table II.
Triples in Table III.
Regions 1, 3 doubles in Table IV
Regions 2, 3 doubles in Table V

CIRCUMSTANCES ALTER CASES

Figure 11

Any given pair of alternative chromatid types occurs in each of the Tables, (I-V), but in each table it represents a different type of crossing over. Thus in terms of an initial cross of *sb* and *bl le c* (or *sb* + + + × + *bl le c*) the ten different combinations of *sb*, *bl*, *le* and *c* represent a different kind of crossing over in each table, as is shown above. The alternative pairs of chromatids are readily derived from the F_1 maternal formula by indicating crossing over by × in the required regions (1, 2, or 3).

one of quadruples. The coefficients of the binomial expansion thus indicate the numbers of combinations possible in multiple crossing over.

In the example under consideration (Table IV) the alternatives carrying *bl* are in all cases lower, presumably because of lower viability. These are listed and totalled in the second column of alternative genotype frequencies. The order of genes on the map is determined from the totals of each pair of alternatives which are listed in column 2. These totals are set down in six columns (Table VII), each column including the four groups of recombinations between two allelic pairs. Each of the six columns of recombinations is headed by the two mutant symbols separated from each other by "&" if they are together in the parental formula or written together, opposite "+", if they are separated in the parental formula. Thus in listing the frequencies to be included as recombinations between any two pairs, it is necessary only to run through the genotypes representing a member of each pair (Table IV, Column 3) and to record the total frequency for that pair (Column 2).

The totals of the recombinations for each two pairs form the basis for making up the linkage map (Figure 12). Three of the gene pairs may be placed at the angles of a triangle with numbers of recombinations indicating lengths of the sides. Of any three pairs, the two having the greatest number of recombinations are farthest apart on the map, except that as this number approaches half the grand total of counts (50 percent recombinations) a limit is reached so that chance fluctuation or viability differences may happen to show slightly more recombinations between genes closer together. In data of Table IV there are 2,290 recombinations between *sb* and *c* and 2,281 between *sb* and *le* among 4,670 grand total. This happens to check with the true arrangement but such a slight difference is unreliable as a guide. In data from +/*sb bl le*

TABLE I. Sons of unmated females *sb bl le c* / + + + +

Cross-over regions	Totals	*bl*+		*bl*		% *bl*	
..	2137	+	1514	*sb bl le c*	623	29.2	Crossover units
1	1529	*sb*	936	*bl le c*	593	38.8	38.1
2	1145	*le c*	693	*sb bl*	452	39.5	30.5
3	346	*c*	234	*sb bl le*	112	32.4	14.2
							Coincidence
1,2	467	*sb le c*	245	*bl*	222	47.5	77.3
1,3	260	*sb c*	149	*bl le*	111	42.7	104.1
2,3	179	*le*	118	*sb bl c*	61	34.1	99.4
1,2,3	85	*sb le*	48	*bl c*	37	43.5	
Totals	6148		3937		2211	36.0	

overs or non-recombinations, words which suffer the double disadvantage of being both cumbersome and negative. Both "parentals" and "originals" have obvious disadvantages. The writer has for many years employed the word *straights* in this connection. Since it is intended merely to designate combinations of certain genes that were in the egg and in the sperms giving rise to the heterozygote regardless of linkage relationships, a single term appears to be adequate. It is used logically as the antonym to recombinations, but in a linkage experiment the non-crossovers are straights for all the genes concerned.

c (Table I) there are 3,113 recombinations between *sb* and *le* and only 3,105 between *sb* and *c* among 6,148 grand total. In data from *bl c/sb le* (Table V) there are 3,235 between *sb* and *le* and only 3,211 between *sb* and *c* among 6,571 grand total. Construction of the map must be based upon the lower frequencies of recombination, if more than one of the frequencies approximates 50 percent of the grand total.

After the order of genes has been determined and the map constructed, the crossover regions for the different alternative pairs of genotypes may be set down (Table IV, Column 1).

Coincidence

For calculating the three ratios of coincidence the triples must be added to each of the three types of doubles. Thus the total 1,2 crossovers are 377; the 1,3 are 223; and the 2,3 are 159. Each of these divided by the grand total gives the ratio of actual two-region crossovers. The corresponding expected two-region crossovers may be obtained from the product of the ratios of the total crossovers in each of the two regions to the grand total. It is not necessary, however, to calculate any of these ratios of actual or expected crossovers in order to obtain the ratios of coincidence. The actual numbers may be set down as in Table VIII for data of Table IV, from which it may be seen that the ratios of coincidence may be most easily obtained by multiplying the total simultaneous crossovers for the two regions by the grand total and dividing by the product of the total crossovers in each of the two regions. In Tables I-VI the percentages of total recombination in each of the three regions are given in the last column in the rows for the singles. These percentages may tentatively be called crossover units although it is recognized that the distances are great enough to include some doubles. Hence the numbers of true crossover units should be somewhat greater than those given. Percentages of coincidence are given in the rows for the doubles.

If it is desired to test the significance of a deviation from unity in the ratio of coincidence, a two-by-two table may be set up and the χ^2 test applied. Table IX shows this for the 1,2 coincidence ratio of Table IV. Since region 3 crossovers are disregarded, the 238 region 3 singles are added to the 1,774 straights to obtain the total 2,012 observed straights for regions 1 and 2 (1 *s*, 2 *s*); the 172 regions 1, 3 doubles to the 1,168 region 1 singles to obtain the total 1,340 observed region 1 recombinations, region 2 straights (1 *r*, 2 *s*); the 108 regions 2,3 doubles to the 833 region 2 singles to obtain the total 941 observed 1 *s*, 2 *r*; and the 51 triples to the 326 regions 1,2 doubles to obtain the total 377 observed 1 *r*,

TABLE II. Sons of unmated females *sb* + / + *bl* / + *le* / + *c*

Cross-over regions	Totals	*bl*+		*bl*		% *bl*	
..	3346	*sb*	2149	*bl le c*	1197	35.8	Crossover units
1	2058	+	1416	*sb bl le c*	642	31.2	35.0
2	1932	*sb le c*	1003	*bl*	929	48.1	33.5
3	840	*sb c*	508	*bl le*	332	39.6	18.8
							Coincidence
1,2	817	*le c*	523	*sb bl*	294	36.0	85.9
1,3	436	*c*	297	*sb bl le*	139	32.0	95.1
2,3	423	*sb le*	219	*bl c*	204	48.2	97.0
1,2,3	190	*le*	127	*sb bl c*	63	33.2	
Tot'ls	10042		6242		3800	38.0	

TABLE III. Sons of unmated females + / + *sb* / *le* / + *bl* / + *c*

Cross-over regions	Totals	*bl*+		*bl*		% *bl*	
..	1370	*le*	773	*sb bl c*	597	43.6	Crossover units
1	866	*sb le*	459	*bl c*	407	47.0	35.8
2	602	*c*	407	*sb bl le*	195	32.4	27.2
3	130	*le c*	70	*sb bl*	60	46.2	8.7
							Coincidence
1,2	233	*sb c*	142	*bl le*	91	39.1	78.2
1,3	83	*sb le c*	45	*bl*	38	45.8	101.6
2,3	55	+	43	*sb bl le c*	12	21.8	98.7
1,2,3	23	*sb*	15	*bl le c*	8	34.8	
Totals	3362		1954		1408	41.9	

TABLE IV. Sons of unmated females + / + *sb* / + *bl* / *le* / + *c*

Cross-over regions	Totals	*bl*+		*bl*		% *bl*	
..	1774	*c*	1382	*sb bl le*	392	22.10	Crossover units
1	1168	*sb c*	678	*bl le*	490	41.95	36.8
2	833	*le*	618	*sb bl c*	215	25.81	28.2
3	238	+	193	*sb bl le c*	45	18.91	12.2
							Coincidence
1,2	326	*sb le*	185	*bl c*	141	43.25	77.8
1,3	172	*sb*	107	*bl le c*	65	37.79	106.6
2,3	108	*le c*	77	*sb bl*	31	28.70	99.0
1,2,3	51	*sb le c*	27	*bl*	24	47.06	
Totals	4670		3267		1403	30.04	

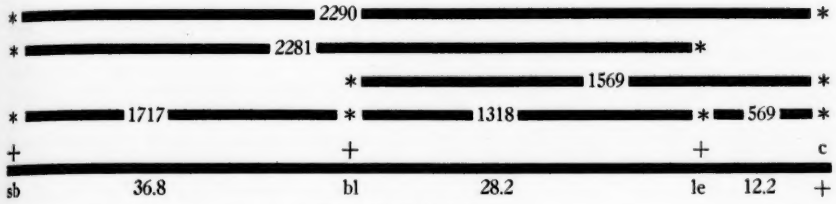
TABLE V. Sons of unmated females + / *bl* / + *sb* / + *le* / + *c*

Cross-over regions	Totals	*bl*+		*bl*		% *bl*	
..	2468	*sb le*	961	*bl c*	1507	61.1	Crossover units
1	1904	*le*	1009	*sb bl c*	895	47.0	41.7
2	1014	*sb c*	493	*bl le*	521	51.4	26.8
3	236	*sb le c*	90	*bl*	146	61.9	9.3
							Coincidence
1,2	575	*c* ..	345	*sb bl le*	230	40.0	86.1
1,3	202	*le c*	104	*sb bl*	98	48.5	101.9
2,3	115	*sb*	55	*bl le c*	60	52.2	105.2
1,2,3	57	+	43	*sb bl le c*	14	24.6	
Totals	6571		3100		3471	52.8	

(from Clark 1943, P. 62.)

TABLE VI. Totals from Tables I - V.

Crossover regions	Averages of percentages	Total frequencies	Based on frequencies Percentages	
..	36.87	11095	36.03	Crossover units
1	25.02	7525	24.44	37.35
2	17.81	5526	17.95	29.97
3	5.30	1790	5.81	13.73
				Coincidence
1,2	7.68	2418	7.89	81.91
1,3	3.56	1153	3.74	98.69
2,3	2.56	880	2.86	101.45
1,2,3	1.18	406	1.32	
Grand total		30793		



THE LINKAGE MAP

Figure 12

Figures between the asterisks indicate total recombinations between indicated genes among 4670, the total sons counted from *c/bl le sb* mothers. Tentative crossover or Morgan units are given between the symbols for the genes. (Based on the data of Table IV.)

2 r. Calculated expectation without interference is based on the marginal totals. Thus the calculated 1 *r*, 2 *r* is $\frac{1{,}318 \times 1{,}717}{4{,}670} = 484.58$. Expectation for the other three cells may be found by subtraction from the marginal totals and since there is one degree of freedom the deviation will be the same (± 107.58) for each cell, but in the opposite direction for the 1 *s*, 2 *s* and 1 *r*, 2 *r* as for the 1 *r*, 2 *s* and 1 *s*, 2 *r*. χ^2 calculated by the usual formula leaves but an infinitesimal chance of agreement with expectation or, in other words, crossing over in region 1 certainly interferes with crossing over in region 2.

In Tables I - V the alternative combinations containing the mutant gene black are listed to the right of those containing its normal allele. The percentages of black among each pair of alternatives are then given (Column 7). The black types are in all cases lower in viability than their alternatives except in four of the pairs shown in Table V. These are the four in which *bl* has segregated opposite to *sb*. In order to study relative viability of the alternative genotypes, the data of Tables I - V were listed and summarized in sixteen groups, each group containing five pairs of frequencies of similar genotypes, one from each of Tables I - V. The lowest viability is shown by *sb bl le* vs. *c* (1,068/3,733), 28.6 percent, and *sb bl le c* vs. + (1,336/4,545), 29.4 percent, and the highest by *bl c* vs. *sb le* (2,296/4,168), 55.1 percent. The quadruple mutant type might be expected to be lowest and in general *sb* in combination with *bl* lowers viability considerably. However, there is evidence that factors, other than the four visibles presumably linked modifiers of viability, play a part. Thus the high viability of *bl c* vs. *sb le* (2,296/4,168), 55.1 percent, is due to the fact that this group of frequencies includes the large straight pair of Table V in which table *bl* shows a very high viability in all combinations, except in the combination of low frequency in which it is a triple crossover, *sb bl le c*. It may be supposed that a "eugenic" selection occurred in Clark's *bl c* stock from which it was necessary to obtain

TABLE VII. Frequencies of recombination arrangements of pairs of mutant genes among sons of unmated females, *c/bl le sb*. (Based on the data of Table IV.)

c bl & +	*c le* & +	*c sb* & +	*bl* & *le*	*bl* & *sb*	*le* & *sb*
833	238	1168	833	1168	1168
238	172	833	326	326	833
326	108	238	108	172	172
172	51	51	51	51	108
1569	569	2290	1318	1717	2281

TABLE VIII. Calculation of coincidence ratios. (Based on the data of Table IV.)

Cross-over regions	Totals	Regions 1,2	Regions 1,3	Regions 2,3
..	1774	$\frac{377}{4670}$	$\frac{223}{4670}$	$\frac{159}{4670}$
1	1168			
2	833	=	=	=
3	238	$\frac{1717}{4670} \times \frac{1318}{4670}$	$\frac{1717}{4670} \times \frac{569}{4670}$	$\frac{1318}{4670} \times \frac{569}{4670}$
1,2	326			
1,3	172	$\frac{377 \times 4670}{1717 \times 1318}$	$\frac{223 \times 4670}{1717 \times 569}$	$\frac{159 \times 4670}{1318 \times 569}$
2,3	108	=	=	=
1,2,3	51			
Total	4670	0.778	1.066	0.990

TABLE IX. Chi²-test of significance of crossover regions 1,2 interference. (Based on the data of Table IV.)

		1 s		1 r	Totals	
2 s	o	2012	o	1340		$\frac{-107.58^2}{2119.58} = 5.46$
	c	2119.58	c	1232.42	3352	
	diff.	−107.58	diff.	107.58		$\frac{107.58^2}{1232.42} = 9.39$
	o	941	o	377		
2 r	c	833.42	c	484.58	1318	$\frac{107.58^2}{833.42} = 13.89$
	diff.	107.58	diff.	−107.58		
Totals		2953		1717	4670	$\frac{-107.58^2}{484.58} = 23.88$
						$\chi^2 = 52.62$

TABLE X. Sons of unmated females $\frac{+ \quad le \quad +}{bl \quad + \quad c}$

(from Clark 1943, Tables 2, 12, 13.)

Cross-over regions	Totals	*bl*⁺		*bl*		% *bl*	
..	14151	*le*	8107	*bl c*	6044	42.7	Crossover units
1	6326	*c*	3735	*bl le*	2591	41.0	30.6
2	2300	*le c*	1218	*bl*	1082	47.0	13.6
							Coincidence
1,2	932	+	579	*bl le c*	353	37.9	94.2
Totals	23709		13639		10070	42.5	

χ^2 for coincidence = 5.559 $P < 0.02$

females for crossing. Correspondingly if genes are introduced from the male side they may be linked with "dysgenic" modifiers since the fathers are usually segregates from heterozygous females and the mutant type has not, therefore, been subjected to such rigid selection. In agreement with this it may be noted that *sb bl* is in all combinations of lower viability in Table IV than in the other Tables (18.9 percent vs. 29.4 percent average for *sb bl le c* vs. +; 28.7 percent vs. 38.9 percent average for *sb bl* vs. *le c;* 22.1 percent vs. 28.6 percent for *sb bl le* vs. *c;* and 25.8 percent vs. 40.9 percent average for *sb bl c* vs. *le*). There is no lowering of viability due to *bl* in Table IV in comparison with similar genotypes of the other Tables when *bl* is separated from *sb*. Therefore it may be supposed that some viability reducer occurred between *sb* and *bl* in this material. In contrast, *sb bl* combinations are not noticeably reduced in Table I in comparison with similar genotypes of the other tables. It may be concluded that while the various combinations of visibles determine differences in viability, there is no constant relationship in this material and that numerous viability modifiers affect the haploid males.

Table VI totals Tables I - V according to crossover regions. In column 2 the percentages of the frequencies of the alternative combinations are averaged so that the five different experiments are given equal weight. Column 3 gives total frequencies for the different regions and column 4 gives percentages of these frequencies out of the grand total of all experiments, 30,793. The crossover and coincidence percentages in column 5 are based on total frequencies. If viability differences among the different genotypes caused appreciable differences in relative frequencies of the different pairs, this might in part be compensated by giving equal weight to the different experiments. Similarity of percentages in columns 2 and 4 and approximate similarity of crossover and coincidence percentages in the different experiments may be taken as evidence that disparity in viability of the different genotypes is compensated to a considerable extent by summing the alternatives.

Evaluation of Results

The new data agree with those of Clark[2] as regards length of the three regions, except that *sb - bl* averages slightly shorter. They also show that there is significant interference of crossing over in *sb - bl* with *bl - le*, but they indicate no interference across *le*. Clark has pointed out the possibility of the spindle fibre being located near *le* and many data indicate lack of interference. However, Clark reported three experiments from *le* females crossed with *bl c* males which were not summarized together and considered in this respect. This has been done in Table X from which it may be seen that coincidence is somewhat less than 100 percent. Application of the χ^2 test shows probability of agreement with random distribution of crossing over to be less than two percent. There is, therefore, a suggestion that some slight interference may be present in some experiments. Considerable variation in crossing over has been noted for the *bl - le* region in different experiments so that differences probably exist between different stocks. Possibly inversions play a part and such would also affect coincidence. Interferenec of *bl - le* with *le - c* might occur if the centromere were at an appreciable distance from *le* in some strains while in strains with close approximation of *le* and the centromere coincidence would be complete.

After conclusion of discussion of four-point linkage and after the students are familiar with the fact that meiosis segregates the four chromatids of each tetrad into the three polar bodies and the reduced egg nucleus, the meaning of alternativeness of the genotype pairs is considered. The question is raised—if *sb*, for example, remains in the egg, does the combination *bl le c* necessarily occur in one of the polar bodies and what then occurs in the other two? This leads to consideration of four-strand crossing-over and the relation of crossing-over to chiasmata.

Literature Cited

1. Bridges, C. B. *Amer. Nat.* 66:572-574. 1932.

2. Clark, Arnold M. *Proc. Pa. Acad. of Sci.* 17:47-64. 1943.

A PEDIGREE OF GENERALIZED LENTIGO

ALAN C. PIPKIN AND SARAH BEDICHEK PIPKIN*
School of Medicine, American University of Beirut, Lebanon

GENERALIZED lentigo (lentigenes) is distinguished from ordinary freckling (ephelides) by the presence of small brownish macules. These appear not only on exposed regions of the body but also on areas which are sheltered from the sun by clothing such as the thighs, abdomen, back, and chest. Ephelides also tend to become paler in the winter, whereas lentiginous spots do not show seasonal variation in shade. Furthermore, according to Ormsby and Montgomery,[3] there is no thickening of the epidermis or proliferation of rete ridges in ephelides which characterizes generalized lentigo.

A thorough study of a case of lentigo was made by Zeisler and Becker.[6] The patient was the only member of her family thus affected, so that no information concerning the possible genetic nature of the abnormality was available. In a case of generalized lentigo reported by Rosen,[5] a brother and a sister of the patient were also said to possess pigmented spots though only in small numbers.

In the present study, generalized lentigo was found in eight individuals belonging to three generations of a family of Maltese-Lebanese extraction.

The propositus of the pedigree, III-4, was covered with dark reddish-brown macules, varying in size from a pin head to a large pea (Figure 14). These spots were most densely concentrated on his face and neck although they were present in large numbers on his arms, dorsal surfaces of hands, chest, back, thighs, and lower legs. Some spots were found on the ears, eyelids, lips, and palms. The upper arms and chest were not often exposed to the sun. As Figure 13 shows, these regions are preceptibly paler in the absence of tanning than the exposed regions. No pigmented spots were found on the conjunctiva. A few of the larger macules were very slightly raised.

A histological examination of a section of skin from the right arm of III-4, the propositus, was made by Dr. Edith E. Sproul, M.D., to whom the authors owe the following description:

"Over half of the section of epidermis is of normal thickness or thinned with few rete pegs. The basal cells contain a very small amount of melanin in fine granules. The dermis beneath this part appears normal. The other half of the section shows an abrupt increase in size of rete pegs with enlargement and increase in number of basal cells. They contain a great abundance of yellow brown nonrefractive pigment resembling melanin but tending to scatter in coarser granules. Similar pigment is prominent scattered superficially through this part of the dermis in individual small rounded or elongated cells. These never form nodules such as observed in the usual nevus. Prussian Blue reaction shows that none of the pigment is hemosiderin."

Lentiginous spots in an older sister (III-1) of the propositus were as intense and as frequent as in the latter (Figure 13). Dark reddish-brown macules covered this girl's face so densely that adjacent spots frequently merged. As in her brother, III-1 possessed spots on the eyelids, lips and palms, as well as on the face, neck, arms and dorsum of hands. She also was affected with pigmented spots on the unexposed regions of chest, back, thighs, and legs. Macules in a younger sister (III-6) whose photograph appears in Figure 13, were paler and not so extensive as in her other two sibs. They are nevertheless clearly a variation of the same abnormality.

The father of the three children with pigmented spots also displayed the anomaly. He and two pigmented brothers were seen by the authors, and a third brother, now dead, was described by them. Photographs of II-1, father of the propositus, and II-5, uncle of the propositus appear in Figure 13. The macules in these individuals appeared as extensive as in the propositus, but they were considerably paler. According to the statement of II-1, his pigmented spots had not altered in color with age. A summary description of all lentiginous members of the pedigree is given in Table I. This shows that the age of onset of lentigo among the different affected members of the

*The authors are indebted to Dr. H. Chaglassian, M.D., for locating the present case, making the biopsy, and for suggestions to Dr. Edith E. Sproul, M.D., for the histological examination, and to Dr. A. Diab, M.D., for the opthalmoscopic examination.

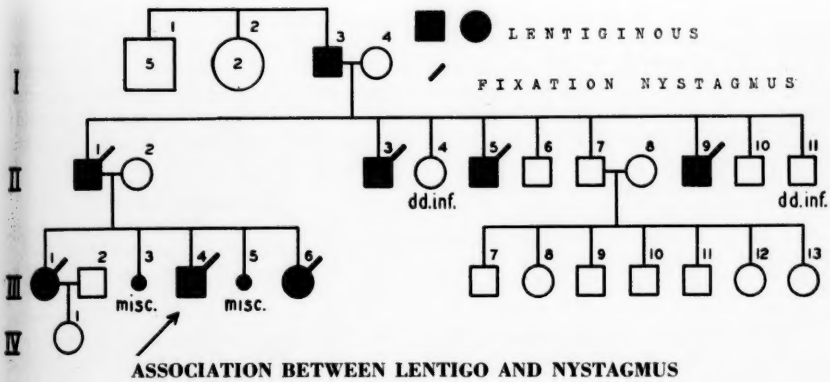
SKIN AND EYES AFFECTED
Figure 13

The individuals shown here are found in generations II and III of the pedigree (Figure 14). Lentigo differs from freckling in the spots being permanent and not limited to the parts of the body exposed to the sun. The oscilation of the eyeball in fixation nystagmus is induced when the individual attempts to follow a finger moving from side to side in front of the eyes. Two views of III-4 are given to show extent of pigmentation and details.

pedigree varied from about four years to ten years of age. The hair of the propositus and his sibs was dark brown; skin, fair, though not so fair as in extremely blond members of the white race. In no affected individuals were erythema, atropic white spots, or telangiectases seen, and only two molelike raised areas were observed. These were on the face of II-1, father of the propositus. Also no pigmented spots were found on the conjunctiva in any member of the pedigree observed by the authors. None of these traits was observed in the following individuals free from the pigmented condition: I-4, II-2, II-7,and all children of II-7.

Upon rubbing the skin of the propositus, III-4, either on regions including spots or on regions without spots, no urticaria resulted.

A pronounced lateral nystagmus was present in the propositus, III-4, and his older sister, III-1. In III-4 the nystagmus resembled that found in negro albinos[4]; that is, it was present continuously. In III-1 the nystagmus was irregular; while sometimes occurring spontaneously, at other times it was not apparent though it could be induced. Strabismus was present in both III-1 and III-4 and it was reported to have been present in II-3, now deceased. An ophthalmoscopic examination of the eye of III-4 made by Dr. A. Diab, M.D., revealed a normal fundus. Other lentiginous members of the pedigree examined, displayed a lateral nystagmus similar to a fixation nystagmus. They developed nystagmus upon following a finger moved from side to side in front of the eyes. The non-lentiginous mem-



ASSOCIATION BETWEEN LENTIGO AND NYSTAGMUS

Figure 14

With one exception, the individuals in this family exhibiting lentigo also have nystagmus. No other record of such an association is found in the literature.

bers were found not to possess a fixation nystagmus such as was present in any lentiginous individuals of the family.* IV-1 was too young to be tested for a fixation nystagmus. Her eyes were apparently normal. No evidence of photophobia was found in any member of the family.

Information regarding the pedigree was obtained from I-4, II-1, II-7, and III-8. The authors examined I-4, II-1, II-5, II-7, II-9, II-10, and all members of generations III and IV. The father of the propositus, II-1, came from the island of Malta, whereas his wife was Lebanese, confirming their claim of no kinship.

There was some doubt in making a diagnosis of generalized lentigo in this pedigree due to the similarity of the symptoms to certain other pigmented conditions. Ephelides was ruled out, although this trait is inherited as an autosomal dominant according to Hammer, as quoted by Cockayne,[1] because in the present case the pigmented spots occur upon unexposed parts of the body as well as on exposed parts, and also because the present case shows increase in size of rete pegs with enlargement and increase in number of basal cells. The histological examination shows that the condition is not the usual type of nevus, nor is it due to a breakdown of hemoglobin since none of the pigment proved to be hemosiderin.

That the pigmented macules might represent an atypical form of Von Recklinghausen's disease was rejected for the reason that seven affected individuals of the pedigree were examined, and none of them exhibited white spots, tumors or pronounced raised areas (with the exception of two molelike structures on II-1). Atypical cases of xeroderma pigmentosa have been described which resemble the case under consideration. However, none of the seven affected members of the present pedigree showed erythema, atrophic white spots, telangiectases or photophobia. Furthermore, xeroderma pigmentosa is inherited as a partially sex-linked recessive abnormality,[2] whereas the present abnormality is inherited as a simple autosomal dominant. Thus in borderline cases a pedigree study may help differentiate atypical cases of xeroderma pigmentosa from generalized lentigo.

TABLE I. Description of generalized lentigo among different members of the pedigree.*

Pedigree number	Age when seen	Age onset macules	Intensity of pigmentation
II-1	51	4	yellowish red
II-5	35	4	yellowish red
II-9	32	6	yellowish red
III-1	26	6	dark reddish
III-4	21	9	dark reddish
III-6	16	10	yellowish red
II-3	d. at 45	5	not seen

* With three exceptions the pigmentation extended without noticeable differences on the face, lips, neck, ears, back of the hands and arms, palms, legs, chest, thighs and back. In the case of three individuals (II-1, II-5, and III-6), there was an absence of macules on the lips and the palms. II-5 had macules on the ears.

*The normal individuals tested were I-4, II-2, II-7, II-8, II-10, III-7, III-8, III-9, III-10, III-11, III-12, and III-13.

No previous description of generalized lentigo records any ocular abnormality associated with the pigmented condition. The nystagmus in the present pedigree which varied considerably among the different lentiginous members, might be dependent upon a gene closely linked to the gene causing lentigo with no crossing over in the present pedigree. On the other hand, nystagmus could be a manifold effect of the same gene causing lentigo. The only other case known to the authors where pigmented spots (presumably freckles) and nystagmus occur together, is in negro albinos and San Blas Indian albinos.

Summary

A pedigree of generalized lentigo with eight affected members in three generations was described which followed autosomal dominant type of inheritance. The extent and intensity of pigmented spots varies among the different affected members of the pedigree. A fixation nystagmus was associated with the lentigo in the present pedigree.

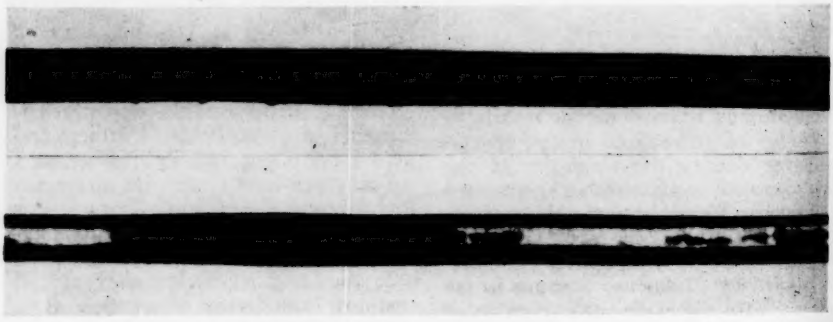
Literature Cited

1. Cockayne, E. A. Inherited Abnormalities of the Skin. Oxford Univ. Press. 1933.
2. Haldane, J. B. S. *Ann. of Eugen.* 7: 28-57. 1936.
3. Ormsby, Oliver S., and H. Montgomery. Diseases of the Skin. Philadelphia. 1943.
4. Pipkin, Alan C., and S. B. Pipkin. *Jour. Hered.* 33:418-427. 1942.
5. Rosen, Isadore. *Archiv. Derm. and Siphilol.* 45:979-980. 1942.
6. Zeisler, Erwin P., and S. William Becker. *Archiv. Derm. and Siphilol.* 33:109-125. 1936.

✦

FOUR GENERATIONS OF RINGED HAIR

Laurence M. Ashley* and Robert S. Jacques



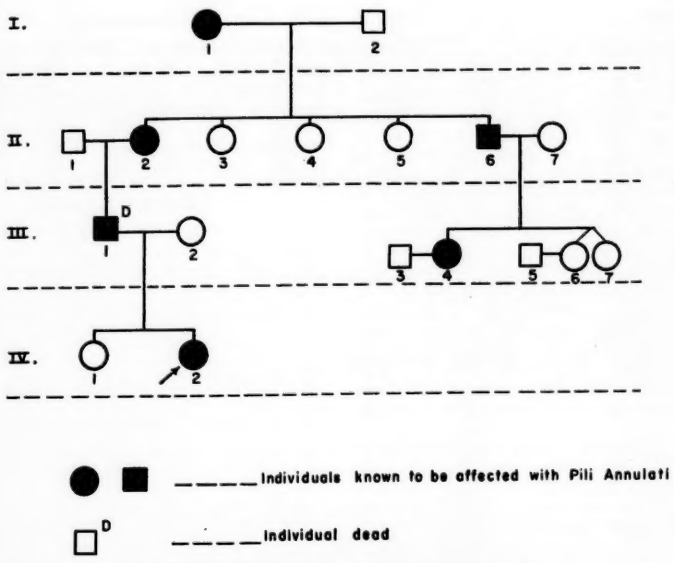
NORMAL AND "RINGED" HAIR

Figure 15

The banded appearance of ringed hair is due to the inclusion of air in the cortex. Ringed hair is weaker than normal hair, and tends to be "self-bobbing."

Pili annulati or Ringelhaare (ringed hair) is one of the rarest inherited anomalies manifest in man. In this condition the hair is normally cylindrical, but is clearly differentiated into light and dark zones which are due to the presence of alternating aggregations of air bubbles in the cortex. Karsch, in 1864, was the first to record ringed hair and gave it the name "pili annulati." Between 1864 and 1922, there were recorded only eighteen other cases, of which fourteen were described in the literature. In that year, Cady and Trotter[2] published a résumé of these cases and also added the histories of three more affected families. Since 1922 instances of ringed hair have been reported by Snell and Foley[7], Joun[5], Erhardt[3], Reyn[6], and Bory and Galy[1]. With the inclusion of the pedigree we are submitting, investigations have revealed the occurence of pili annulati in 27 different families.

*Professor of Zoology, Walla Walla College, College Place, Wash.



FOUR GENERATIONS OF RINGED HAIR
Figure 16

A sample of ringed hair under gross examination appears the same as would normal hair except for the alternating light and dark bands. These contrasting areas present sharply defined segments. Microscopic study reveals no marked histological deviation from normal hair other than the contrasting light and dark segments.

The aspect presented by pili annulati when observed en masse, is that of hair which appears lusterless, greying, sandy-colored or speckled. In some cases the hair may be shorter than normal due to the fact that it tends to break off at the light, gas-filled, segment.

Karsch in his description of ringed hair endeavored to explain the light areas in the normally dark hair as white pigment areas. Speiss in 1859, however, offered the theory that the light bands could be attributed to air or gas bubbles which would indicate a lack of any pigment. In support of Speiss we also have the conclusion of Cady and Trotter. Based on the refractive behavior of ringed hair examined under transmitted and reflected illumination, these authors concluded that: "Appearance of pili annulati is caused by gas-filled interstices in the cortex and medulla of the hair. The appearance is not caused by lack of pigment or pigment atrophy."

The first indication that pili annulati might be a result of hereditary transmission was advanced by Galloway[4] when he reported the cases of two brothers. In all cases recorded where the pedigrees of the families concerned can be traced, this condition follows a pattern of dominant or irregular dominant inheritance. The pedigree of our case is an example of dominant inheritance.

In presenting this pedigree of pili annulati we wish to thank Miss Catherine Chonkich who first called our attention to this case and who also submitted the pedigree to us. We have investigated this family and have verified the pertinent facts to our own satisfaction.

The propositus, (IV-2), had always been under the impression that her hair was sandy colored, until it was brought to her attention that her hair had a banded appearance. Upon further observation there could be plainly seen with the unaided eye the alternating light and dark bands peculiar to pili annulati. All the hair of her head was affected. Because the action of combing tended to break the hair at the air-filled zones, her hair was never longer than six inches. It did not break off at the roots. Additional investigation bore out the fact that she had been born with banded hair and that it had been transmitted through her father, (III-1). III-4, II-6, and I-1 were similarly affected. It occurs in all generations and about half of each generation is affected: the mode of inheritance is simple dominance.

Some authors consider this condition to be synonymous with monilithrix or beaded hair. From our investigation we have come to consider these two conditions as being decidedly different. A few of these differences will be given. Monilithrix hair gives the appearance of being beaded or having constrictions in the

hair at regular intervals, while pili annulati is cylindrical throughout with no change in the hair circumference at any point. Weber and Axhausen[8] point out the difference by stating, "The appearance is (speaking of monilithrix), however, quite different than that of "ringed hair" (Ringelhaare) in which there are no true constrictions . . ." Also, follicular keratosis (keratosis pilaris) usually accompanies monilithrix which is seldom true in the case of pili annulati. The affects of pili annulati are confined to the hairs of the head for the most part, while monilithrix is usually manifest in the hair of other parts of the body as well as the head. In cases of monilithrix the hair of the head is sparse, while in cases of pili annulati there is usually a normal growth of hair over the head. The hair has a tendency to break off at the roots in monilithrix, but in pili annulati the hair may vary in length from the normal to that of only an inch or so in length.

Cady and Trotter affiirm that there is no evidence of pathology connected with pili annulati. It is presumed that many cases of pili annulati go undetected because there are so few accompanying disorders.

Summary

A pedigree has been offered presenting four generations of pili annulati in which simple dominance is the obvious mode of inheritance. A brief review of the literature has been presented. The contrasting characteristics of monilithrix and pili annulati have been discussed.

Literature Cited

1. Bory and Galy. *Ann.* de *Derm.* et de *Syph.* 3:295. 1943.
2. Cady, D. D. and M. Trotter. *Arch. Derm. Syph.* 6:301-317. 1922.
3. Erhardt, S. *Munch. Med. Wochens.* 79:949-951. 1932.
4. Galloway, J. *Brit. Jour. Derm.* 8:437. 1896.
5. Joun, M. *Dermatologia* (Basel) 86:117-122. 1942.
6. Reyn, A. *Brit. Jour. Derm.* & *Syph.* XLVI:168. 1934.
7. Snell, G. D. and F. Foley. *Jour. Hered.* 23:155-157. 1932.
8. Weber, F. P. and J. Axhausen. *Brit. Jour. Derm.* & *Syph.* 39:62. 1927.

✦

Human Races and the Soviets

To the Editor:

THE November 1949 number of the Journal of Heredity contains a translation of statements of Professor A. N. Studitski which are so weird that it is difficult to take them seriously. Many of them are as ludicrous as some of the footnotes of German commentators on Shakespeare's plays. My *Human Ancestry* is quoted to the effect that "Polynesians, Melanesians and Negroes emerged as human races at a comparatively late period." There is nothing particularly wrong with that statement as it stands, but he goes on to make it appear that this means that "the black races quite recently emerged *out of a simian state*"! Perversion of meaning could hardly go further. I say in fact (p. 337), "Originating in India or Java, . . . the proto-Polynesians are believed to have launched into the Pacific from Java about the time of Christ." No one who reads my book could regard Professor Studitski's statement as anything less than an intentional misrepresentation. He speaks of "reactionary delirium" which, however, is not mine but his.

The rest of his diatribe is unworthy of further notice, but I may take this opportunity to correct certain other statements that have been made. He says (p. 308), "Darwin hotly defended the idea of the unity of the human race." Darwin's views on human races were fully discussed in Chapter XI of my book. He did not "hotly defend" anything. That was not his way. Instead, he put forward a careful and well-balanced discussion, based on the knowledge of his time, from which I have made a number of quotations.

This paragon of fairmindedness announces that eugenics is a "reactionary perversion of science." He decries the sterilization of imbeciles, but his country sends millions to their death in labor camps under subhuman conditions and has the effrontery to label as "democracy" a condition in which "liquidation" is the penalty for failure to toe the line regarding the latest developments in Communist dogma. He refers to need of *Lebensraum* as an excuse for aggression by the "German fascists," but the Russian Communists have committed aggression on the whole of Eastern Europe without even this excuse. He says "The world will never forget the crimes committed by German fascism." Why should it forget Communist crimes against millions of their own people as well as nearly the whole population of Eastern Europe, which they are endeavoring to enslave.

It is clear that when a tyranny has so much to hide they must catch at straws in their efforts to hoodwink the people over whom their tyranny is exerted. But truth will out, and even a slave state cannot ultimately suppress it. They use the shibboleth that "all men are equal" to fasten the shackles more securely on their slaves. One hopes that is politically true, at least outside of Communist rule, but every biologist knows that biologically no two men are alike in their desires, aptitudes or achievements.

R. Ruggles Gates

Biological Laboratories,
Harvard University